TRANSVERSE MAGNETIC WAVES IN MYELINATED NERVES

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Abstract- **The structure of myelinated axons is quite similar to that of a high-loss coaxial cable. An electromagnetic analysis of TM waves shows that distributed effects cannot be neglected. The attenuation and phase constants are obtained as a function of frequency. Predicted finite wave delay in the internodal segment approaches measurements.**
Keywords **- Nerves, models, elctromagnetism, waves, velocity, delay, attenuation, myelin**

I. INTRODUCTION

Electric stimulation of nervous system can restore motor functions [1]. Electrical properties of nerves are ususally described by means of a cable model [2], [10]. This model is frequently simplified, and each internodal segment is modeled by a lumped impedance. This can be a series resistor [3]-[7], but sometimes parallel capacitive impedances are added [27]. A lumped resistor is unable to model a finite conduction velocity in the internodal segment, whereas the presence of capacitors modelling the myelin sheath can explain delays [27]. Despite this fact, resistor models are more frequently used, and the delay is usually assigned only to nonlinearities in Ranvier nodes [15].

The existence of travelling waves in internodal segments could linearly explain, at least partially, the measured conduction speed [14]. The objective of this paper is to determine whether under any circumstances a distributed circuit [28] covering the effects of both the capacitive and resistive effects would be more suitable than a lumped model. To answer this question we shall analyze an internodal segment through which a transverse magnetic (TM) wave is guided [20], [26]. This approach links with the spatially distributed description of electric and magnetic fields found in literature [7], [11]-[13], [18], [19].

From an electromagnetic point of view, lumped element models are obtained as a quasi-static approximation [20]. Clark *et al.* [21] calculate the wave number k as

$$k = \omega\sqrt{\mu_0\varepsilon\left(1+\sigma / j\omega\varepsilon\right)} \tag{1}$$

For axoplasm at 1kHz with conductivity 5 mhos/m, the resultant wave number approximates 0.198 rad/m. In 10 cm., the phase shift is less than 0.02 radian or 1.46º, so in [21] conclude the field is quasi-static. This conclussion was later adopted explicitly [22] or implicitly in lumped element models [3]-[7], [27]. We shall show that the waves are not defined by k, but rather by the propagation constant h, and that quasi-static approximations don't apply always, specially for relatively high frequencies.

II. METHODOLOGY

For sake of simplicity, we shall consider an infinitely long segment, surrounded by an infinite extracellular medium. Even though the results will not be the same found in physiological situations, qualitative conclusions might be useful to gain understanding into nerve conduction. The structure is quite similar to a high-loss coaxial cable (fig. 1). Medium 1 is the axoplasm, medium 2 is the myelin sheath, and medium 3 is the extracellular fluid.

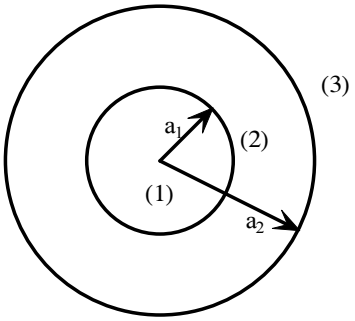

Fig. 1. Myelinated nerve coaxial model.
Axon radium is a_1 and fiber radium is a_2.

We shall assume isotropic, homogeneous and linear media [11]-[13],[16]-[18], although the physiologic medium in which the nerve is embedded is neither isotropic [8]-[9] nor homogeneous [4]. The myelin sheath is sometimes considered as a perfect insulator [3], [7], [18]. We don't assume this hypothesis [27]. All media will be generic with permitivity ε_i, permeability μ_0, and conductivity σ_i, where subscript i refers to medium 1, 2 or 3. Let the media be charged due to present ions. When these ions come into the axon at Ranvier nodes, they diffuse [23], and thus a diffusion current appears. Therefore, the current density **J** at any point designed by **r** in any media, when an eletric field **E** dependent on frequency ω appears can be expressed as [24]:

$$\mathbf{J}(\mathbf{r},\omega) = \sigma\mathbf{E}(\mathbf{r},\omega) - \sum_i D_i \nabla\rho_i(\mathbf{r},\omega) \tag{2}$$

where the sum extends to all present ions ($Na^+;K^+;Cl^-$...) [23] and D_i is the diffusion constant of ion i. The ionic diffusion would exist even if the ions were uncharged particles [24].

The magnetic field intensity **H** is solenoidal and may, therefore, be derived from the curl of a suitable Hertzian vector potential function $\mathbf{\Pi}_e$ [25], [26]:

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Abstract

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$$\mathbf{H} = j\omega\varepsilon_{eq}\nabla\times\mathbf{\Pi}_e \tag{3}$$

where

$$\varepsilon_{eq} = \varepsilon_0\varepsilon_r - j\frac{\sigma}{\omega} \tag{4}$$

Substitution of (3) into Faraday's law and subsequent integration leads to

$$\mathbf{E} = k^2\mathbf{\Pi}_e + \nabla\phi \tag{5}$$

where k was defined in (1) and ϕ is an arbitrary scalar potential function. Using (1) in Ampère-Maxwell's law,

$$\nabla\times\mathbf{H} = j\omega\varepsilon_{eq}\mathbf{E} - \sum_i D_i\nabla\rho_i \tag{6}$$

Sustituting (3) into (6) we find that

$$\nabla\times\nabla\times\mathbf{H} = k^2\mathbf{\Pi}_e + \nabla\phi - \sum_i \frac{D_i\nabla\rho_i}{j\omega\varepsilon_{eq}} \tag{7}$$

Since ϕ is yet an arbitrary function, we propose the following gauge:

$$\phi = \nabla\cdot\mathbf{\Pi}_e + \sum_i \frac{D_i\rho_i}{j\omega\varepsilon_{eq}} \tag{8}$$

Hence an homogeneous wave equation is obtained for $\mathbf{\Pi}_e$. Let's adopt a cylindrical coordinates system axially centered in the axon (r,θ,z). The wave equation reduces to

$$\nabla^2\mathbf{\Pi}_e + k^2\mathbf{\Pi}_e = 0 \tag{9}$$

Despite the existence of a difussion current, the election of the gauge given by (8) makes this equation to be the same as if the media were uncharged. Transverse magnetic modes may be derived from $\mathbf{\Pi}_e = \pi_e\mathbf{z}$, thus assuming $\mathbf{\Pi}_e$ have no other components but the axial. The solution to wave equation (9) is [25]:

$$\mathbf{\Pi}_e = \sum_{n=0}^{\infty}\frac{1}{\lambda_n^2}a_n\psi_n\mathbf{z} \tag{10}$$

where a_n is an amplitude constant and ψ_n is a is a space and frequency dependent function given by

$$\psi_n = Z_n(\lambda_n r)e^{-jn\theta}e^{-jh_n z} \tag{11}$$

in which $Z_n(\lambda_n r)$ is a generic Bessel function of order n. By definition,

$$\lambda_n = \sqrt{k^2 - h_n^2} \tag{12}$$

In these equations, $h_n = \beta_n - j\alpha_n$ is the propagation constant, inherently different to k. Phase constant is β_n (rad/m) and α_n is the attenuation constant (Np/m). We shall proof that the thickness of the axon myelin sheath is much less than the wavelength of physiological signals, so that the asymmetric modes (dependent on θ, and thus with $n \neq 0$) are evanescent [25] and attenuate quickly ($\beta_n=0$). Therefore, the main mode has $n=0$ and the fields will correspondingly be denoted $\mathbf{E}_0$ and $\mathbf{H}_0$. The electric field can be obtained using (8) and (10) in (5):

$$E_{0r} = -\frac{jh_0}{\lambda_0^2}a_0\frac{\partial\psi_0}{\partial r} + j\omega\mu\frac{1}{k^2}\sum_i D_i\frac{\partial\rho_i}{\partial r} \tag{13}$$

$$E_{0\theta} = j\omega\mu\frac{1}{k^2}\sum_i D_i\frac{1}{r}\frac{\partial\rho_i}{\partial\theta} \tag{14}$$

$$E_{0z} = a_0\psi_0 + j\omega\mu\frac{1}{k^2}\sum_i D_i\frac{\partial\rho_i}{\partial z} \tag{15}$$

The magnetic field is found from (3):

$$H_{0r} = 0 \tag{16}$$

$$H_{0\theta} = -\frac{jk^2}{\mu\omega\lambda^2}a_0\frac{\partial\psi_0}{\partial r} \tag{17}$$

$$H_{0z} = 0 \tag{18}$$

Note the absence of axial component in the magnetic field in this propagation mode, despite the diffusion current. This is therefore a TM mode [26]. At $r=0$ the field must be finite so $Z_0^{(1)} = J_0$, with J_0 the Bessel function of the first kind and order 0. In the external fluid, the field can not increase with distance and should behave like a travelling wave; therefore $Z_0^{(3)} = H_0^{(1)}$, where H_0 is the Hankel function. In the myelin sheath, $Z_0^{(2)} = J_0 + c_0N_0$, with N_0 the Bessel function of the second kind and order 0, and c_0 a constant to be determined.

Boundary conditions for the continuity of E_z and H_θ at $r=a_1$ y $r=a_2$ lead to an inhomogeneous equations set for $a_0^{(1)}$, $a_0^{(2)}$, $c_0a_0^{(2)}$ and $a_0^{(3)}$. For the system to have more than a single solution, and allow waveguiding happen independently of charge gradient, the determinant of this set of equations must vanish. The determinantal equation is the same that can be found for uncharged media [25]:

$$\frac{\lambda_2 k_1^2 J_1(\lambda_2 a_1) N_0(\lambda_2 a_1) - \lambda_1 k_2^2 J_0(\lambda_1 a_1) N_1(\lambda_2 a_1)}{\lambda_2 k_1^2 J_1(\lambda_1 a_1) j_0(\lambda_2 a_1) - \lambda_1 k_2^2 J_0(\lambda_1 a_1) J_1(\lambda_2 a_1)} - \frac{\lambda_2 k_3^2 H_1^{(1)}(\lambda_3 a_2) N_0(\lambda_2 a_1) - \lambda_3 k_2^2 H_0^{(1)}(\lambda_3 a_2) N_1(\lambda_2 a_2)}{\lambda_2 k_3^2 H_1^{(1)}(\lambda_3 a_2) J_0(\lambda_2 a_1) - \lambda_3 k_2^2 H_0^{(1)}(\lambda_3 a_2) J_1(\lambda_2 a_2)} = 0 \quad (19)$$

The roots of equation (9) together with (12) are the propagation constants h_{0m}, with $m=1,2,3....$

III. RESULTS

The electrical properties of media shown in Fig. 1 can be found in Table I. We are interested only in h_{01}, since higher modes present stronger attenuation. Figs. 2 and 3 show the the phase constant β and the attenuation constant α as a function of frequency. Frequency values have been chosen so as to cover the whole spectrum of physiological signals.

Signal velocity can be calculated as ω/β (fig. 4). Wavelenghts are determined as $2\pi/\beta$ (see Table II).

TABLE I
ELECTRICAL PROPERTIES OF MYELINATED NERVE MEDIA

Parameter	Symbol	Value	Unit	Reference
Axoplasm Conductivity	σ_1	0.909	S/m	[29]
Myelin Conductivity	σ_2	3.45e-6	S/m	[30]
External Conductivity	σ_3	0.333	S/m	[31]
Axoplasm Permitivity	ε_1	ε_0	F/m	
Myelin Permitivity[a]	ε_2	$25'5\varepsilon_0$	F/m	
External Permitivity	ε_3	ε_0	F/m	
Axon Radium[aa]	a_1	6.2	μm	
Fiber Radium	a_2	10	μm	[23]

[a]Derived from coaxial capacity 5e-3 μF/cm^2 [10], [24]
[aa]Using $2a_1 = 0.8 \times 2a_2 - 1.8$ [32]

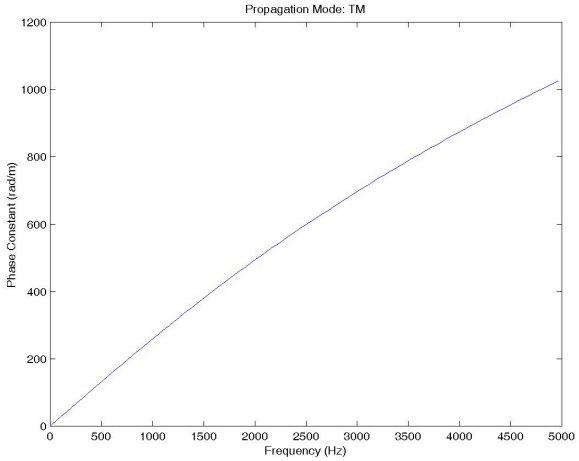

Fig. 2. Phase constant as a function of frequency

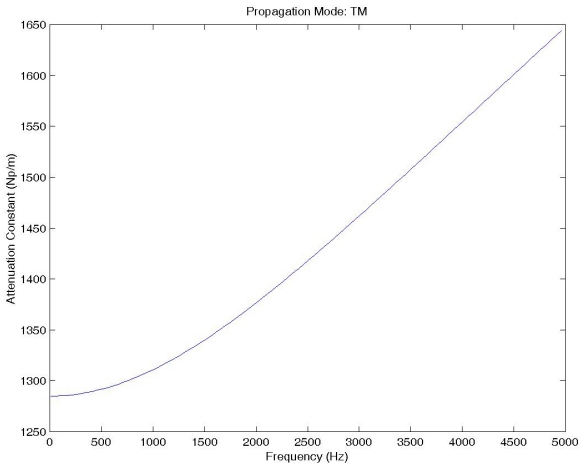

Fig. 3. Attenuation constant as a function of frequency.

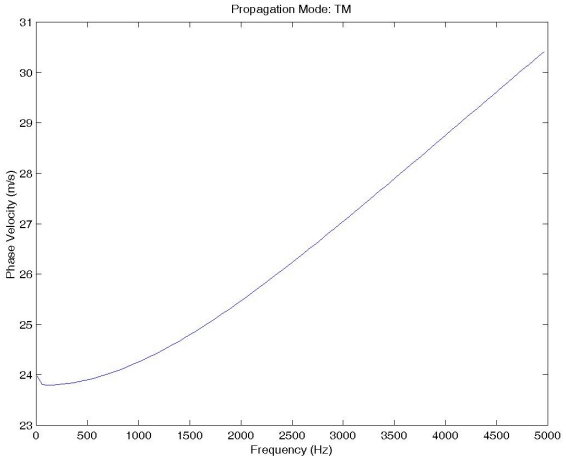

Fig. 4. Wave velocity as a function of frequency.

IV. DISCUSSION

Phase constant is, at 1kHz, higher than 200rad/m (125.6º in 10cm). This value is far away from 0.198 rad/m pointed out by Clark *et al.* [21], [22] and shows the need for a distributed parameter model. Note that, as one could expect, this is more true as frequency increases. Therefore, the nerve behaviour is not always quasi-static. Wavenumber and propagation constant are the same when the wave is TEM [26], but we are dealing with TM modes. Attenuation constant is more or less constant at frequencies below 250 Hz, and then increases linearly. For a typical internodal distance of 1 mm [23], low frequency signals attenuate up to 0.28 times their value, despite their frequency. At higher frequencies, attenuation grows approximately at a 3 dB/decade rate.

The order of magnitude of concerning wavelengths are a thousand times greater than the thickness of the myelin sheath, validating a previous assumption.

Signal velocity is independent of frequency up to 500 Hz. Values are similar to those measured in [33] (25-41 m/s), [14]. Therefore delay is not only due to Ranvier nodes, but also to internodal segments. This result is in agreement with [27], but no we have used no lumped element.

Equation (2) is an extension of Ohm's law. Lumped element circuits neglect the diffusion current, taking into account only the drift current due to the electric field. But ionic diffusion would be present even in absence of axial electric field.

TABLE II
WAVELENGTHS IN MYELINATED NERVES

Frequency (Hz)	Wavelength (mm)
50	396
100	216
500	46
1000	24
2500	10
5000	6

V. CONCLUSION

Whereas lumped circuit models are much easier to deal with, the quasi-static hypothesis doesn't hold for relatively high frequencies and a distributed model might be more adequate. TM waves linearly model attenuation and non-zero delay in the internodal segment, and could help to gain more insight into neural artificial stimulation problems. Further research is required to analyze other propagation modes.

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